REVIEW

Ventricular Arrhythmias Originating from the Papillary Muscles of the Left Ventricle in the Structurally Normal Heart and the Role of Catheter Ablation

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Abstract

Ventricular arrhythmias arising from the left ventricular papillary muscles, having a right bundle branch block (RBBB) QRS morphology on the electrocardiogram, account for approximately 15% of arrhythmias in a structurally normal heart and have usually a benign prognosis. The mitral valve usually has two papillary muscles, anterolateral and posteromedial, with the latter one being always more arrhythmogenic. These arrhythmias are not typically inducible by programmed ventricular or atrial stimulation, suggesting a non-reentrant mechanism. Ventricular arrhythmias originated from the papillary muscles should be distinguished from other idiopathic left ventricular arrhythmias such as fascicular or mitral annular arrhythmias, which also have a RBBB pattern. Catheter ablation in these cases is always challenging and the recurrence risk is higher compared to other idiopathic ventricular arrhythmias. ***Rhythmos 2021;16(1):95-98.***

Keywords: ventricular arrhythmias; papillary muscles; catheter ablation; electroanatomical mapping; activation mapping; pace mapping

Abbreviations: APM: anterolateral papillary muscle, CA: catheter ablation, ICE = intracardiac echocardiography; LV: left ventricle, PICM: tachycardia-induced cardiomyopathy, PMs: papillary muscles, PPM: posteromedial papillary muscle, PVCs: premature ventricular contractions, RF = radiofrequency, VT: ventricular tachycardia.

Introduction

The papillary muscles (PMs) in the left ventricle (LV) are a source of ventricular arrhythmias (VAs) both in structurally normal and abnormal hearts. Ventricular arrhythmias can occur either as isolated premature ventricular contractions (PVCs), or ventricular tachycardia (VT).[1,2] Catheter ablation (CA) has been described as an effective treatment for these arrhythmias, although radiofrequency energy delivery at PMs has been associated with poor manipulation and catheter stability compared with other locations.

Anatomy of Left Ventricle Papillary Muscles

The PMs demonstrate unique anatomy characteristics in both the left and the right ventricle. They are among the thickest endocardial structures, and fibers on the papillary muscles have separations, likely contributing to anisotropy.[3] The mitral valve usually has two papillary muscles, posteromedial and anterolateral. The posteromedial papillary muscle (PPM) is perfused by either the right coronary or the circumflex artery, while perfusion to the anterolateral papillary muscle (APM) is supplied both from the left anterior descending and the circumflex arteries. Therefore, the posteromedial PM is more vulnerable to ischemic injury than the anterolateral muscle. The APM has mostly a single head and originates from the anterolateral LV wall and provides chordae to the anterolateral half of the anterior and posterior mitral leaflets. The PPM originates from the infero-septal LV wall and provides chordae to the posteromedial half of both leaflets.[4,5] The PPM is a more common location of origin of ventricular ectopy than the APM. A rich subendocardial network of Purkinje fibers exists in PPMs which is usually connected to the myocytes near the PM base.[6] This close relationship between the Purkinje fiber and PPMs has led to some difficulties to distinguish arrhythmias from the two structures.

Mechanism of arrhythmia and clinical presentation

Idiopathic VAs from the PMs are usually sensitive to catecholamines and are not inducible by programmed ventricular or atrial stimulation, thus suggesting a non-reentrant mechanism. [7,8] A triggered activity or abnormal automaticity mechanism is further supported by the lack of fragmented potentials at the sites of local activation during mapping of these arrhythmias. Triggered activity or abnormal automaticity mechanism may be promoted by decreased Purkinje-ventricular coupling at the PPM that affects the electrical loading of the Purkinje cells by the neighboring myocardial cells.[9] This close relationship between the Purkinje fiber and PMs makes sometimes difficult the differential diagnosis between arrhythmias originating from these two structures. The PMs may also be involved in post-infarction VAs due to scar related reentrant mechanism.[10]

Most commonly, PM-related arrhythmia is presented as premature ventricular complexes (PVCs) and/or non-sustained ventricular tachycardia (VT).[11] Less commonly patients exhibit sustained VT which could be induced by exercise. A high burden of PVCs can lead to PVC-induced cardiomyopathy (PICM) with a benign course after elimination of PVCs with catheter ablation (CA). [12]

Electrocardiographic recognition

Patients with VAs originating from the LV PMs, have a right bundle branch (RBBB) morphology of the QRS complex in lead V1. More specifically, VAs from the PPM usually have a RBBB morphology with a left superior axis, (positive in leads I and aVL, negative in aVR, negative in II and III) and precordial transition at V3-V5. On the other hand, arrhythmias from the APM often have an inferior axis, similarly to arrhythmias from the left anterior fascicle and the anterior part of the mitral annulus.[13] It is sometimes difficult to distinguish between VAs originating from PMs and fascicles. Compared with fascicular arrhythmias, the QRS is wider in PM VAs (150±15 vs. 127±11ms; *p=0.001*).[14] An rsR´ pattern in lead V1 is characteristic of fascicular arrhythmias which have also a small q wave in either lead I or aVL (qR or qRs).[15] Absence of q waves in the lateral leads is recorded in VAs originating from the PPMs since they are lying more laterally and septal depolarization occurs later in the QRS complex. An algorithm, with approximately 90% sensitivity and specificity, has been published using a QRS width greater than 130 milliseconds as a cutoff value to diagnose the origin of VAs between fascicle and PMs.[16] Mitral annular VAs often display positive concordance pattern, due to the left ventricle basal location, which is not addressed in PM or fascicular VAs.[16]

The Role of Catheter Ablation

Idiopathic VAs arising from the LV PMs account for approximately 15% of idiopathic LV arrhythmias referred for CA.[8] The expert consensus statement recommendation on CA of VAs published in 2019 gives a class I (level of evidence B) indication for CA in patients with frequent PVCs or recurrent monomorphic VT originating from PMs. Generally, these VAs often require several radiofrequency (RF) energy applications for successful ablation. [17] The recurrence risk is reported to be higher than what is reported for many other idiopathic VA sites, and repeat procedures are required in approximately 30% of patients. High burden of PVCs, defined as percentage >10-20% of the total QRS/24h has been related to the presence of PVC-induced cardiomyopathy.[18]

Catheter ablation of PM VAs is always challenging because of the following: (a) lack of good contact and stability of ablation catheter, (b) limited efficacy of pace mapping, (c) frequent need for ablation at different sides of PM due to change in the exit from the PM into the LV.[8] Access to LV PMs could be either retrograde via the aorta or trans-septal.[19] Usually, the PPM and the medial aspect of the APM are best approached with a retrograde access, while the lateral aspect of the APM after trans-septal puncture (**Fig. 1**). The latter approach is preferred also in patients with prosthetic aortic valve and/or peripheral artery disease.[19]

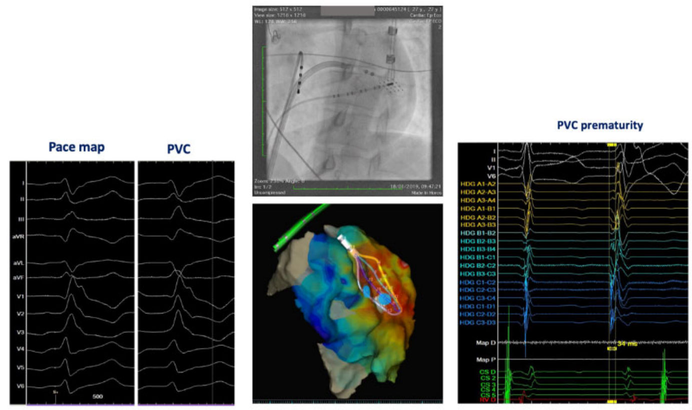

Figure 1. Ablation of APM PVCs via a trans-septal approach using an Agilis™ steerable sheath with a large curve (Abbott, St. Paul, MN). Activation mapping with the EnSite Precision™ Cardiac Mapping System and the Advisor™ HD Grid Mapping Catheter, Sensor Enabled™ (Abbott, St. Paul, MN) shows site with activation -34 ms pre-QRS at the base of APM. At this site pacemapping was identical to clinical PVC (12/12 matching leads) and radiofrequency ablation with 50W with the TactiCath™ Contact Force Ablation Catheter, Sensor Enabled™, resulted in completely disappearance of PVCs. *(Henry Dunant Hospital Center EP Lab)*

Activation mapping of the clinical PVCs or VT requires the presence of spontaneous PVCs and is performed with electroanatomical mapping systems using either the ablation catheter or multielectrode mapping catheters. In the absence of spontaneous ectopy, induction is achieved with burst pacing and/or isoproterenol infusion in patients without structural heart disease. Sites with the earliest activation suggest close proximity to the arrhythmia focus and ablation at these sites is usually successful resulting in arrhythmia elimination. The recorded signal on the tip of the ablation catheter at the site of arrhythmia origin is usually sharp with QS pattern on the unipolar electrogram.[19] In approximately 40% of cases, a sharp Purkinje potential can be recorded at the successful ablation site, which suggests involvement of the Purkinje system in the arrhythmogenic mechanism.[13,20] Pace-mapping is always necessary when PVCs are infrequent or secondary to confirm the site of ablation. However, pace-mapping is associated with inconsistent results given that it correlates only with the exit point of the arrhythmia and not with the site of origin. Yamada et al. demonstrated that despite excellent pace-mapping, the radiofrequency energy delivered to the site of best match failed to terminate the PVC in 42% of patients. Furthermore, in this study, approximately 50% of patients with PM VAs exhibited variable QRS morphologies spontaneously, after the initial ablation lesions, or both. In approximately 80% of these patients, radiofrequency lesions on both sides of the PMs were required to eliminate all different QRS morphologies.[13]

High power radiofrequency energy application is delivered (usually up to 50 Watts) using an open irrigated contact force ablation catheter, targeting an impedance drop of 10-15 Ohms or 10% of the starting impedance value. It is crucial to stop the delivery of the energy when a sudden impedance drop >15 Ohms is recorded in order to avoid a steam pop and LV free wall perforation. This is because the catheter ablation can be "entrapped" between the base of the PM and its intersection with the LV free wall. Stability can be improved by pacing faster especially if the patient is bradycardic due to continuous bigeminy.[19]

Cryoablation is an alternative when radiofrequency ablation fails due to poor contact and stability. The advantages of cryoablation include better contact regardless of the mobility of PM. On the other hand, the most important disadvantages are the poor maneuverability, the reduced lesion depth and the lack of integration with the electroanatomic mapping systems. Rivera et al published a retrospective series comparing 12 patients who underwent cryoablation with 9 patients who underwent radiofrequency ablation. Acute procedural success was reported in all 12 patients who underwent cryoablation and in 7/9 patients undergoing RF ablation (100% vs. 78%; P=0.08). With success defined as a ≥ 50% reduction in VAs, at a mean follow-up of 360 days without antiarrhythmic drugs, the cryoablation group had a 100% success rate versus a 56% success rate at 87 days follow-up in the radiofrequency group.[21]

Intracardiac echo (ICE) is a useful tool to ensure adequate catheter-tissue contact and correct orientation of the catheter tip during mapping and ablation. Moreover, ICE imaging may identify increased echogenicity in the PMs, suggesting scar regions which potentially could be low voltage areas with abnormal electrograms, such as late potentials. With the proper manipulation and rotation of the ICE catheter into the LV, both APM and PPM can be well imaged, providing safety and efficacy in the ablation procedure. [19]

Recently published data, about ablation of PM VAs, showed that acute suppress of VAs was achieved in 95% of the patients and the long-term freedom was about 91%. The use of contact force technology did not influence the efficacy of ablation procedures. On the other hand, the use of ICE in all cases resulted in significantly shorter procedure, fluoroscopy and RF ablation times. As regarding the safety of CA, no significant valvular dysfunction immediately post-procedure according to ICE and as measured by echocardiography at a mean follow-up of 36 months, was reported in 60 LV PMs ablation procedures.

Conclusion

Idiopathic VAs arising from the LV PMs account for approximately 15% of idiopathic LV arrhythmias referred for CA and are characterized by frequent PVCs or recurrent monomorphic VT with a catecholamine-sensitive focal mechanism. Despite their generally benign prognosis, CA may be required for symptomatic patients or those with PVC- or tachycardia-induced cardiomyopathy. Catheter ablation in these patients is always challenging with variable short-term and long-term success rates, because of poor contact and stability of the catheter. The use of ICE is always essential to resolve these problems, while on the other hand, cryoablation is an alternative to radiofrequency ablation with better results regarding tissue contact.

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